Original Articles.

EYE-PARALYSES.*

BY JOHN AMORY JEFFRIES, M.D.

[This paper, which Dr. Jeffries had nearly finished at the time of his death, has been put in my hands to prepare for publication. Although I knew something of his intentions in regard to it, a study of the paper itself and of the mass of material which he had collected on the subject has convinced me of the difficulty of completing the task as he would have done it. With the exception of a few verbal changes, indicated in brackets, I have done little except to prepare a bibliography from the notes which Dr. Jeffries left, and to indicate the references to illustrative cases. Not having the original articles at hand and depending almost wholly upon these notes I fear that the classification of the cases, which I have tried to make, is not always correct, and that it might not agree with Dr. Jeffries' greater knowledge. I have added Bleuler's diagram, which Dr. Jeffries had copied, probably with the intention of inserting it. — P. C. K.]

PROBABLY every practitioner has at times been in doubt as to the nature of a case of eye-paralysis. The whole question is involved in practical difficulties. A patient complains of double vision; and it is evident, unless the case be one of monocular diplopia, that at least one of the twelve extrinsic muscles of the eyes is paretic. In theory, the changes in the relative position of the images in different parts of the visual field should lead to an accurate diagnosis; in practice, owing to the complex results produced when a number of muscles are affected, and to the stupidity of patients, the result is often far from satisfactory. Even when the eye distinctly lags in following an object, it may be difficult to determine the condition of the oblique muscles. When, as sometimes happens, the patient fixes with the paretic eye, there is danger that the trouble may be assigned to the wrong eye. The paretic eye is used for fixation when it has the best sight or when it is specially trained, as in the case of the right eye in surveyors. It is therefore desirable to make a systematic study of every case of eye-paralysis with all the methods at our command.

For this purpose it is necessary to have a pair of spectacles carrying plane glasses, one colorless, the other red, so that the color of the image may indicate to which eye it belongs. After assuring one's self that the diplopia is not of monocular origin, by having the patient look first with one eye and then with the other, the whole field should be tested with some bright object, as a candle-light, and the result noted in a chart-like manner. The relative position of the images, their parallelism or obliquity, the distance between them, and which is red, should be determined for at least three points directly in front of the patient, and for two tiers on each side representing half-way and full lateral vision. When, as is often the case, vision is impaired as well as the motions of the eye-ball, care must be taken lest monocular vision due to lack of vision in the peripheral portion of one retina be mistaken for proof of parallel vision. After this, near vision should be tested in the same way.

The double images had better be studied first, since they require close attention on the part of the patient and error is not easily recognized by the examiner.

It must be borne in mind that lack of double vision does not prove the absence of paralysis. One of the images may be disregarded, lost, or there may be but one, as in cases of conjugate paralysis, where there is constant parallelism of vision even though motion to one side is absolutely lost.

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This is hardly the place to go into the details of the position of the images. They have been so fully worked out and figured in the various works on the eye and nervous system as to be familiar to all. Those interested in the difficult subject of double paralysis of the superior obliques will find the subject elaborated in a recent article by Pflüger.[114] This paralysis is probably best recognized by the perimetic method.

Next the motions of the eyes should be observed when following some object, as a finger, a piece of paper, or the like. With practice very slight deviations from the normal can be recognized in this way. Not only the parallelism of vision, but the range of motion should be noted; also the position of the lid, and whether it follows the pupil in looking down or not. Very good motor charts can be made by Landolt's method in cases where the defect of motion is considerable. This consists simply in substituting a card with letters on it for the plain moving card of a perimeter, and after having instructed the patient to follow it as long as he can and still read the letters, slowly move the card from the centre to the periphery. The point where distinct vision ceases should be noted on any of the charts used in plotting out the field of vision. A muscular defect shows itself as a limitation in that part of the field towards which the weak muscle should have moved the eye. The method is based on the fact that the letters cannot be read unless their images fall on or very near the visual axis of the eye. Those who have not used this method, might think that it was entirely dependent on the promptness and accuracy of the patient's replies. This is not the case, as the reflections from the cornea render it very easy for the physician to recognize the faintest motion of the eye. Of course, this method fails entirely in those relatively rare cases where the paralysis is dependent upon binocular vision.

Several other factors remain to be determined which are more physiological in their nature; thus, it should be noted if there is any difference in the motion of the eye when its mate is covered. A quite marked or even complete paralysis may vanish at once if the other eye be covered, while in others the paralysis is only apparent after the other eye is closed. Secondary deviation of the sound eye should be noted by placing a screen between the eye to be tested and the object gazed upon, and then removing it; if the deviation exists, the eye will be seen to fly back a few degrees as the object comes into view. It is due to the correlated muscles of the sound eye rotating the eye beyond the line of parallel vision, from sympathy in the effort required to fix the object with the paretic one. Hence, the direction of the motion is that in which the other eye is weak.

In making this test the screen should be held sufficiently near the eye to prevent fixation being performed by it and yet in such a way the eye can be seen by the observer. The readjustment of vision is at times very quick, and the process may be finished before it can be noted.

False projection, if well developed, is also a factor of considerable value. This — the failure properly to locate objects in space — is held to be due to our judging of the relation of objects to ourselves not primarily by the position of their images on our retinæ, but by the state of contraction of the extrinsic muscles of the eye. We first determine the direction of the objects falling in the line of distinct vision by the eye muscles and

then locate the rest of the field by means of the relation of the peripheral images to the centre of the field. As a result of this, if a small object — say a pin stuck into a table — is fixed with the paretic eye and the patient be instructed to pick it up quickly the hand will miss the object and go too far in the direction towards which the motion of the eye is weak. Of course, in making this test, the other eye should be closed, the motion made rapidly, and the pin placed so as to involve the use of the affected muscles. When the paretic eye is closed, no false projection occurs, and the hand goes straight to the object. When both eyes are open, false projection in the opposite direction may result from overaction of the muscles of the healthy eye. When both eyes are affected, the last two tests often give most perplexing results.

Besides the above, the reaction of the pupil to light, near vision, and sensations from the skin should be noted as well as the action of the ciliary muscles. At the time of, or shortly after, the occurrence of a paralysis, the eye is apt to deviate in the opposite direction, owing to the contraction of the antagonists, the same as in other parts of the body. But, in spite of this, the eye is often able to move in the direction of the paralyzed muscle as far as the mid-point.

The result of the examinations gives us a knowledge of the muscles paralyzed, the degree of paralysis, and the conditions under which it occurs. These facts have still to be correlated with what we know of the physiology of the eye, both normal and pathological, as well as with the results of clinical observation and the post-mortem examination before a full diagnosis can be made, and the physician be truly said to understand the case. A full diagnosis calls for a determination not only of the parts paralyzed, but also of the portions of the nervous system affected, and the nature of the affecting process. Fortunately for the patient, the second part of the diagnosis has little to do with treatment; only in surgical cases is an exact knowledge of the seat of disease called for by therapy.

The physiology of the muscles of the eye is by no means a simple problem; not only do we have to explain how the muscles bring the eyes into the proper position, but also to trace out the track through the nervous system by which this is affected. In the normal inactive state, where vision may be said to be in abeyance, the eyes rest in a mid-position and with nearly parallel visual axes. I say nearly, because on several occasions I have thought that the eye did not correspond by a few degrees, the same as is common in the blind. Now, if an object to one side of the axis attract attention, the eyes are at once turned so that their axes are brought to bear upon it. This is done by the equal and synchronous action of the internal rectus of one eye and the external rectus of the other, and if the object is not on a level with the eyes, by the action of the muscles turning the eye up or down (superior rectus and inferior oblique or the reverse). This process, lateral conjugate deviation, has attracted much attention, and yet we do not know how it is brought about. Starting with the experiments of Mott and Schaefer [95, 90, 130] and others, we know that this motion is produced by stimulation of the middle portion of the frontal lobe just in front of the head area, the eyes turning to the opposite side. Again, stimulation in the visual field in the occipital lobe causes the same motion, but in an opposite direction — that is, the eyes turn to the side stimulated. Lastly, if the seat in the occipital region where the eyes are turned to one side and that in the frontal region where the eyes are turned to the other are stimulated synchronously with the same current, the motion induced by the frontal area prevails over that of the occipital.

Over and above this, conjugate motion is liable to be induced by stimulation almost anywhere in the cortex. Such conjugate paralysis or deviation has been well recognized since the thesis of Prevost [118] and is one of the most constant early symptoms following almost any sudden gross lesion of the cerebrum. During the comatose period, if there be any, the eyes will be found turned as if looking to the sound side. When consciousness returns, the eyes may still continue in their former position, or assume the normal one. If still turned to one side, a true paralysis may exist, or the position may simply be one of selection, the eyes being able to follow an object well to the opposite side. In either case the trouble usually vanishes in the course of a few hours or weeks, and the eyes return to a normal state. Since in pontine trouble the paralysis is crossed with that of the limbs, the symptom is at times of value in locating the lesion.

In a small proportion of the cases the deviation remains constant, as a permanent paralysis, indicating that the motor region or fibres have been actually injured and not simply disturbed in their functions. Efforts have been made to use this symptom for purposes of localization, but so far with poor success. Thus Wernicke [164] holds that such a paralysis coming on with a shock without loss of consciousness points to disease of the lower temporal region of the opposite side, and cites a few cases beside his own. They are, however, far from satisfactory.

There is a small number of cases where disease of the frontal convolutions has been accompanied by disturbance or paralysis of the eyes of such nature as to suggest some close connection with the lesion, but they are far too few and vague to justify any conclusions, though seeming to tally well with experiments on animals.

As we do not know whence the motions of the eyes spring, it goes without saying that we do not know the course of the fibres conveying the impulse to the nuclei at the base of the brain. They probably pass down in the anterior part of the internal capsule and thence, via the pyramidal tracts, to near the nuclei of the third nerve where they cross. Perlia [111] describes a set of fibres which, coming from the crus, turn and pass back along the râphe and then enter the nuclei from their free surface; these seem to be best explained as being the lower end of the cerebral fibres. The fibres to the sixth nuclei also probably cross somewhere in the same region, since in pontine trouble paralysis of the sixth nerve is always on the same side as the lesion. The sixth nucleus is placed under the eminentia teres in the angle formed by the knee of the facial nerve. Its root fibres pass down and out; the course of its cerebral fibres is not known.

Conjugate deviation in cases of pontine disease is of far more diagnostic value than when springing from disease higher up, and has, since Foville [88] first called attention to the subject, offered material for much discussion and many theories. In 1885 Bleuler [8] collected the cases and made a very material contribution to our knowledge of the subject; recently I have been at some pains to go over the literature and have been able nearly to double the number of cases with autopsy

collected by Bleuler. Even a brief citation of these would swell this article far beyond all limits, nor in view of Bleuler's article is it necessary; but as they show several points of importance, a close summary is required. Owing to the internal recti being supplied by the third nerves, while the external recti receive their fibres from the sixth pair, lesions in the pons are liable to split up conjugate motion into its separate elements.

The following different states are found to exist:

(1) Complete paralysis of one external rectus and the crossed internal rectus, so that neither muscle is capable of any motion, the eyes looking forward or to the other side according to the state of the opposite pair of muscles. In these cases the eyes turn readily to the opposite side, up or down, but stop at the mid-line as if transfixed.

(2) Precisely the same state as in the first condition except that the internal rectus acts perfectly for near or convergent vision.

(3) When both eyes are open, the eye with the paralyzed internal rectus will not turn in for objects on the other side of the nose, but will if the other eye be covered.

(4) The internal rectus will not act in conjugate, but will in near vision: the external rectus being normal.

(5) Though devoid of any signs of conjugate paralysis we must consider simple nuclear sixth nerve paralysis as an element of conjugate paralysis.

[Dr. Jeffries told me that he had omitted to speak fully of the cases in this first class, in which the conjugate paralysis was complete. I find in his notes references to eleven cases, which seem to belong here: those of Leyden,[74] Bernhardt,[6] Ballet,[1] Broadbent,[13] Desnos,[20] Meyer,[84] Foville,[38] Garel,[39] Wernicke,[165] Mills,[88] and Webber.[161]]

In all these cases there were destructive lesions which, as far as can be judged from the report, involved either a considerable area in the pons above the nucleus of the sixth nerve or involved the pons immediately below or in front of the sixth nucleus on the paralyzed side.

In the second group of ten cases [Kahler and Pick,[97] Férčol,[32] Vinantiis,[159] Mills,[89] Hofstetter,[61] Grasset,[47] Poubin,[117] Blocq and Guinon,[9] Finny,[37] Graux,[49]] eight showed a lesion just below or at the eminentia teres. It was in most cases small or at least did not effect much tissue in this region. In the other two, those of Mills[89] and Hofstetter,[61] the lesion was a good-sized tumor in the upper quarter of the pons. The symptoms in the first case do not appear to have been in any way peculiar. In Hofstetter's case there was simply a paresis of conjugate motion and no deviation to the opposite side.

I have only found five cases [Bleuler[8] (two), Spitzka,[144] Quioc,[119] Samelsohn,[126]] where it is clearly stated that the eye with the paralyzed internal rectus turned, as in conjugate motion, to the other side when the other eye was covered. The question is how this comes about. To all appearances the suppression of the eye with the paralyzed external rectus restores power in the other, as if its weakness were due to sympathy and not to actual paralysis; but such an explanation not only fails to explain, but stands in marked contrast to peripheral paralysis of the sixth nerve where this action does not occur. There is nothing peculiar in the autopsies; in two there were tumors of moderate size below the sixth nucleus. In Bleuler's[8] two cases the tubercles, though diffuse, especially attacked the sixth nucleus. In Spitzka's[144] the tumor, though extensive, apparently spared the floor of the ventricle along the râphe.

The only way out of the difficulty is to assume that the apparent conjugate motion in monocular vision is in reality a convergent motion substituted in its place. In this case we might expect the covered eye also to turn in and the pupils to contract. If either occurred, it would be fair to hold the point demonstrated. Unfortunately, in none of the cases has either been noted. But there are cases without autopsy, evidently of the same nature, in which this has been found to be the case. Also the obverse is known to occur in paralysis of convergence. If this view be correct, this group is really indentical with the preceding.

There are three cases which apparently belong to the fourth group. Köchlin[71] reports the case of a boy three years old who after a month's sickness developed a decided deviation of the right eye to the right, and left facial paralysis. The tongue was projected straight; the pupils were equal; the limbs normal and the mind clear; pulse 112. After the paralysis had existed for a month, meningeal symptoms developed, and the child died. At the autopsy the only possible cause found for the paralysis was a tubercle adhering by its base to the floor of the fourth ventricle on the left side at the level of the angle formed by cerebellar peduncles. The region of the third nerve nuclei was normal.

Hunnius[62] reports the case of a man forty years old who had apoplexy. Two days later, up and down motion of the eyes was normal. There was absolute loss of power in the left external rectus, and the two internal recti; convergence and binocular vision could only be executed with difficulty (!); left conjugate motion lost; left eye in mid-position; right turned out forty degrees. Spinal myosis: marked dysarthria; right hemiplegia. Paralysis of right facial, the upper branch being least affected, and right hypoglossus. Head carried turned to the right, but can be turned anywhere. Mind clear, vision good, no nystagmus. By the fifth day the left eye had turned in a little, and the right was not turned so far out, only parallel to left. No other change. Death from pneumonia on the twelfth day. All the cranial nerves normal; softening in the left half of the pons beginning back of its anterior limit and most extensive in the middle. Part of the sixth root apparently implicated, but not the nucleus. Pyramidal tract entirely destroyed.

In the third case reported by Schutz[152] a woman forty-eight years old, after two weeks of weakness, was suddenly seized with paralysis and difficulty of speech, without loss of consciousness. On the fourth day there was diffuse headache, stertorous respiration, rapid pulse, but no œdema. She understood well, but spoke with difficulty. The head was freely moved, there was ptosis on the right, and the left eye did not turn in and down with the right; otherwise, motions of eyes normal. The pupils were small, the left angle of the mouth paretic, the tongue turned to the left. There was slight rigidity of all four limbs, and the right were paralyzed. At autopsy the vessels were found contorted, the ventricles enlarged, the ependyma thickened, and a pair of small cysts the size of peas in the thalami. Also in the upper half of the pons an irregular hæmorrhage, larger than a hazel-nut, in the region of the left seventh nucleus, which spared the

lower parts on the left and part of the right half of the pons.

In the first case Gubler[50] has already given the interpretation accepted by me; the second case belongs, as far as loss of left conjugate motion is concerned, to the second group. It is only the internal rectus of the left eye which is here of interest. This acted in convergence and not in conjugate motion. The presumption is that the fibres of the third nerve system, affected in association with the system of the sixth nerve in conjugate motion, were here affected alone; but the autopsies are too vague to aid in plotting out the position of the fibres.

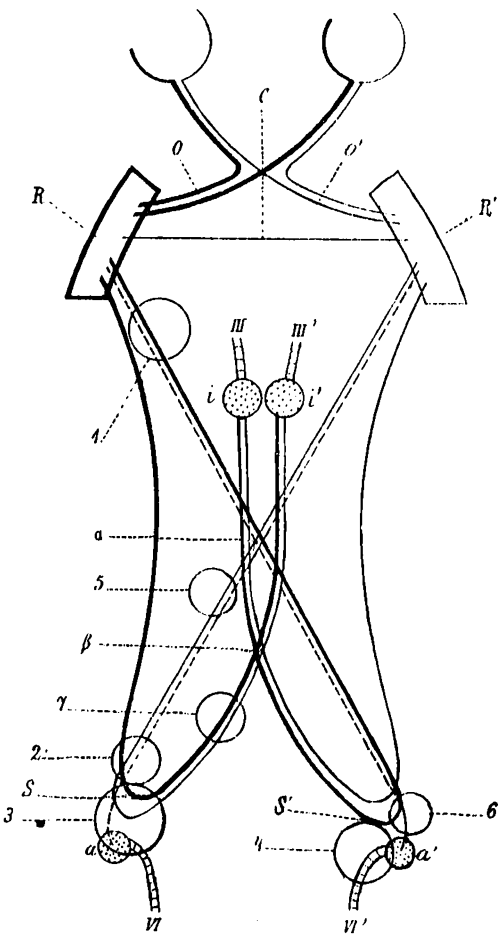
There is one case (Eichhorst[26]) of simple deviation of the eyes and no paralysis due to a lesion in the pons. A man forty-seven years old and a syphilitic, fell in coma after a few hours of moderate pain in the head. On return of consciousness there was right hemiplegia, motor aphasia, relaxation of right side of palate, right facial paralysis, dysphagia. The face and eyes were both turned to the right, but could be moved freely in any direction. There was also a tendency to lie on the right side. The pupils were much contracted and the light reflexes lost. Sensorium free. Death occurred on the eighth day. A clot was found in the anterior part of the basilar artery, and the pons was very soft in the anterior part of the left half. The softening extending nearly to the floor of the fourth ventrical. The cerebellar peduncles were normal. A figure of the basal surface of the pons shows that the softening involved the lower half of the left crus.

To my mind, this is not a case of conjugate paralysis but one of forced rotation to the right, a symptom frequently associated with conjugate paralysis. There was no paralysis of the eyes. All the cranial nerves affected were on the same side as the hemiplegia, and the figure in spite of the text shows the lesions to be in the crus.

Since a central paralysis of the sixth nerve is an essential part of a conjugate paralysis, they are best considered in this connection. Bleuler[8] has classed a case reported by Grasset[47] among those of conjugate paralysis, which to my mind belongs to this group. The head, eyes, and body turned to the right, but the right eye could be turned to the left, while the left could not. Autopsy showed several small sarcomas, two of these being in the left half of the pons. This case seems to be in reality one of conjugate deviation of the eyes to the right and paralysis of the left sixth nerve.

My collection of simple paralyses of the sixth nerve dependent upon injury in the pons offers nothing peculiar in their character. There does not seem to be any point of distinction in action between the different parts of this system. In all, if not from the first, there have been before long other symptoms pointing to the pons. Some cases have begun as conjugate paralysis, and then become simple sixth paralysis; in others the obverse has been the case. In the first group the lesion has been a haemorrhage at the eminentia teres. I have myself seen the two states alternate from time to time. Though there is no autopsy, a brief summary is given, as the case is of some interest.*

Considerable ingenuity has been spent upon the explanation of the mechanism of conjugate paralysis, and various theories evolved. Most of these involve either the existence of a special nucleus beside the sixth nucleus for conjugate motion or fibres arising from the sixth nucleus and ending somewhere in the line for the internal rectus, either in the nucleus of the third nerve or the nerve itself. No one has pointed out the nucleus barring Gowers,[48] who suggests the olivary body, but as this is healthy in many of the cases it needs no consideration. The various fibre tracts have been pointed out by one or another, but not in such a way as to convince the doubter. None of these hypotheses stand in harmony with the facts.



Explanation of Figure (from Bleuler).

O O′, Optic tracts.
R R′, Cortical centres.
a a′, Abducens nuclei.
i i′, Internal rectus nuclei.
S S′, Point where the internal rectus fibres bend.

1, Lesion producing Prévost's symptom.
2, 3, Lesions producing paralysis of one abducens and the opposite internal rectus for all movements.
4, Lesion producing paralysis of one abducens and weakness of the opposite internal rectus for all movements.
5, Paralysis of left abducens, absolute defect of right internal rectus for associated lateral movements, weakness of both internal recti for convergence.
6, Paralysis of abducens and inactivity of opposite internal rectus for lateral movements only.
7, Total paralysis of opposite internal rectus.

α, Double abducens paralysis.
β, Paralysis of convergence.

Bleuler[8] has advanced a theory which was compatible with the facts up to his time of writing, and holds good for the more recent cases. According to this, the fibres run direct from the cortex to their respective root nuclei, but there are two sets of fibres for the in-

* No record of this case is given.

ternal rectus, one for conjugate vision and one for near vision; the fibres run down as separate bundles below their nucleus close to the sixth nucleus, and then turn back and cross to their nucleus. There is no physiological objection to this theory, and it coincides well with the results of pathological investigations — indeed, it was built up from the best summary there is on the subject. It does demand the two sets of fibres, something not held to exist in any other part. On the whole I believe it to be the best working theory we have. The only other way of looking at it seems to be that there is but one set of fibres, these going close to the sixth nucleus, but that the impulses descending from above in conjugate and near vision are different, and that the conjugate impulse is more easily interrupted than the near vision impulse; the nearest analogy being the different results obtained by the stimulation of a nerve as shown by Hooper and Bowditch, and the selection of the extensor muscles in most hemiplegias. Any such theory of course ignores the idea that each muscle is represented by certain nerve fibres, cells and areas, which transmit the force after the style of a complex system of bell cords, — an idea which seems in silence to be creeping into neurology, but does not tally well with complete recovery after resection of a nerve. One can hardly imagine that in these cases the cells in the cord establish their old relations with the muscles. If not, and the pull cord theory be correct, utter confusion should result when any motion is attempted.

Whatever the theory may be, the practical point remains that all cases of partial conjugate paralysis so far reported have depended upon lesions of the pons.

(To be continued.)

REMARKS ON THE RADICAL TREATMENT OF CERTAIN CASES OF GRAVE CONCEALED ACCIDENTAL HÆMORRHAGE.[1]

BY MALCOLM STORER, M.D., BOSTON.

WHILE a concealed accidental hæmorrhage is one of the rarest of the emergencies that can arise in obstetrical practice, it is, of course, liable to occur in the hands of any one of us at any time, and when present, the condition is so grave and the mortality so appalling, even with the best of treatment, that it may be worth while to give the history of a case showing a method of treatment, which, though apparently somewhat heroic, may prove to be, under certain conditions, the one giving most promise of saving the mother. My excuse for presenting this paper is the novelty of the operation and the possibility of its otherwise not coming before you, occurring as it did, in Dublin.

That a concealed hæmorrhage is rare is undoubted, but the small number of cases reported may be partly due to non-recognition of the condition, and to a natural dislike to publish fatal cases.

The total number of cases that I have been able to collect is 153. In 1860, Braxton Hicks [2] reported 23 cases. To these Goodell [3] added 84. I have been able to add 46.[4] While it is not pretended that this is a complete list of all the cases reported, it is sufficiently so to form an idea of the frequency of the condition and to base a few statistics upon.

Dr. Henry C. Coe, of New York, having given notice that he is engaged upon an elaborate paper on the subject, I shall use these cases only to bring out a few points connected with the results of treatment.

From these cases it appears that of 126 children 94 per cent. are known to have been still-born or to have died shortly after birth; while of 152 mothers 71 died, or 46.7 per cent. This mortality is, in many cases, due to non-recognition of the accident and consequent non-treatment. In by far the greater number of the cases the trouble was not recognized until there had been a large loss of blood. Of 63 cases left to themselves (mostly not recognized), in which if anything was done it was merely to give anodynes and stimulants, 64 per cent. died, while of 79 cases treated only 29 per cent. died. After excluding from the 47 cases in which the woman died undelivered, 16 in which the hæmorrhage was so sudden and severe that the patient was practically dead before anything could be done, 13 at least of the remaining 31 probably could have been saved, if proper treatment had been thought of.

It may be worth while, in view of the rarity of the accident, to run through the ordinary symptoms. A concealed hæmorrhage should be suspected, if, during labor, prostration with pallor, nausea, etc., appears, out of all proportion to any external hæmorrhage that may be present, not forgetting that the respiration may become sighing before the pulse is of necessity affected; or should the membranes remain tense as if the uterus were in a state of tonic spasm; or should labor pains cease or grow weak without other cause; or should they be replaced by a dull, constant ache above the pubes; or should it be noticed that the os is dilating without apparent labor pains. The suspicion would be increased if the patient were a multipara in the latter months of pregnancy, and if there were an antecedent injury or fright. An asymmetrical enlargement of the uterus appearing and increasing under the eye of the physician would give certainty. Some writers claim that it is especially liable to occur in feeble or albuminuric women, but this is very questionable.

In these cases the diagnosis is comparatively easy, and the treatment laid down in the books fairly satisfactory, and not especially difficult to carry out, the woman already being in labor, and the cervix, if undilated, probably soft and yielding.

The treatment advised is as follows (Lusk): (1) Subcutaneous injection of ergot; (2) Dilatation of the os with Barnes' dilators; (3) Rupture of the membranes; and (4) version.

[1] Read before the Section for Gynæcology and Obstetrics of the Suffolk District Medical Society, February 20, 1892.

[2] Obstetrical Transactions, 1860, xi, 34.

[3] American Journal of Obstetrics, 1869, xi, 281.

[4] 1. Sinclair and Johnston, Practical Midwifery, 358, Case 29. 2. *Ibid.*, Case 28. 3. Goodell, Journal of Obstetrics, 1871, iv, 333. 4. Weatherly, British Medical Journal, 1878, ii, 284. 5. Marcailhou d'Aymerie, Jour. de Med. et de Phar. de l'Algerie, 79, iv. 6. Lusk, Journal of Obstetrics, 1880, 877. 7. Dowden, Medical and Surgical Reporter, xlvii, 697. 8. Atchinson, Nashville Journal of Medicine and Surgery, 1880, N. S., xxv, 6. 9, 10. Gillfillan, Proceedings of Medical Society of King's County, Brooklyn, 1881, vi, iii. 11. Mundé, Journal of Obstetrics, 1881, 652. 12, 13. Barnes, E. J., Lancet, 1881, ii, 1038. 14. Macan, Dublin Journal Medical Sciences, 1882, 445. 15. Johnson, *Ibid.* 16. Kidd, *Ibid.*, p. 442. 17. Horne, *Ibid.*, p. 439. 18, 19. Wey, Medical Record, N. Y., 1884, 146. 20. Caldwell, Chicago Medical Journal, 1885, i, 291. 21. Cummins, Daniell's Texas Medical Journal, 1885, 350. 22. Maxwell, *Ibid.*, p. 352. 23. Hudra, *Ibid.* 24. Doering, *Ibid.*, p. 210. 25. Reynolds, Boston Medical and Surgical Journal, 1885, cxiv, 463. 26, 27. Anderson, Pacific Medical and Surgical Journal, 1887, xxx, 217. 28. Park, Journal of American Medical Association, 1887, 124. 29. McDavitt, *Ibid.*, p. 144. 30. Squires, New York Medical Record, 1888, xxxiii, 13. 31. Herman, Obstetrical Journal of Great Britain, 1888, viii, 87. 32-34. Jaggard, Philadelphia Medical News, 1889, 600. 35-38. Kortright, Brooklyn Medical Journal, 1890, iv, 646. 39. Longaker, Annals of Gynæcology and Pediatry, 1890, iv, 506. 40, 41. Ribl, *Ibid.*, p. 507. 42. Hirst, *Ibid.*, 43, 44. Price, *Ibid.*, p. 510. 45. Coe, American Journal of Obstetrics, 1891, 152. 46. Bagot, Dublin Journal of Medical Sciences, 1891, 225.